	Aspirin sensitive	Aspirin resistant	p
Platelet volume (fl)	8.78±0.26	8.82±0.30	0.92
Reticulated platelet (%)	8.4±0.52	8.6±0.76	0.82
Serup P selectin (ng/ml)	42.6±4.29	42.9±4.75	0.97
Platelet P selectin (%)	11.1±1.0	9.5±1.5	0.35
Serum thrombopoietin (pg/ml)	130.6±11.3	319.9±97.8	0.01

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C(-260)T polymorphism in the promoter of CD 14 gene is not associated with myocardial infarction in the tunisian population

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Introduction: Recent finding suggest that inflammation plays a role in atherosclerosis and its acute complications. Several known mechanisms may play at least a partial role in this process. One of the most likely mechanisms involves lipopolysaccharide (LPS) and its receptor, CD14. The C(-260)T single nucleotide polymorphism in the promoter region of the CD14 receptor gene has been reported to be associated with a higher risk of MI. Others studies, however, have not corroborated these findings. Considering the contradictory results, the aim of the present study was to investigate the possible association between the CD14 C(-260)T polymorphism and the risk of MI in the Tunisian population.
Material and Methods: A total of 333 Tunisian patients with MI and 345 healthy controls were included in the study. Genotyping was performed by polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) analysis.

Results: The frequency of TT homozygous genotype for the CD14 C(-260)T polymorphism was 24.9% in MI patients and 24.0% in the control group. However, the genotype distribution and allele frequencies were not significantly different between MI and controls subjects. Moreover, the odds ratio for MI associated with the T allele failed to reach statistical significance (OR=1.08; 95% CI: 0.86 - 1.34; P=NS).
Conclusion: These results do not support the hypothesis that the C-260T polymorphism of CD14 gene contributes to the genetic susceptibility to MI in the Tunisian population studied.

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Association of rs 2781666 G/T polymorphism of arginase 1 gene with myocardial infarction in the Tunisian population

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Introduction: Arginase I (ARG I) is the final enzyme of the urea cycle that converts L-arginine to urea and ornithine. Emerging evidence have identified ARG I as a critical regulator for nitric oxide (NO) production via nitric oxide synthase (NOS).
Therefore upregulation of ARG I inhibit NOS-mediated NO and may contribute to endothelial dysfunction. In addition, pathophysiological role of ARG I on vascular disease have been extensively documented, and some recent studies support a role for ARG I in the development and complications of coronary artery disease (CAD). The aim of the present study is to investigate the possible association between rs 2781666 G/T polymorphism of ARG I gene and myocardial infarction (MI) in the Tunisian population.

Methods: In a case-control study, a total of 321 patients with MI and 436 controls were included. The rs 2781666 G/T polymorphism of ARG I was determined by PCR-RFLP analysis.
Results: Patients with MI had significantly higher frequency of TT genotype compared to controls (10.3 % vs 6.7 %; OR (95 % CI), 2.05 (1.19 - 3.52), p=0.009). The MI patients showed higher frequency of T allele compared to the controls (0.32 vs 0.23, OR (95 % CI), 1.58(1.25 - 2.00), p<0.001). The association between rs 2781666 G/T polymorphism of ARG I gene and MI was no longer significant after adjustment for other well established risk factors.

Conclusion: Our results revealed a significant but not independent association between rs2781666 G/T polymorphism of ARG I gene and (MI) in the Tunisian population.

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Lack of association between the -420C>G genetic variant in the resistin locus and myocardial infarction in the Tunisian population

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Objective: Resistin is a polypeptide (hormone) that is specifically secreted from adipocytes. It plays an important role in communication between adiposity and insulin resistance and it has been linked to the pathogenesis of atherosclerosis. Recently, -420C>G, a variant located in the promoter region of the resistin gene (RETN) (rs1862513) was identified. The aim of this study was to investigate the association between this polymorphism and the risk of myocardial infarction (MI) in the Tunisian population.

Design and methods: A total of 787 unrelated male Tunisian subjects including 455 healthy controls and 332 patients who had survived a first MI were prospectively recruited. Standard definitions and criteria for MI diagnosis were employed. Blood samples were obtained after an overnight fast. Serum glucose, TC and TG were measured by standardized enzymatic methods using commercial kits (Roche Diagnostics, Mannheim, Germany) on a Hitachi 912 analyzer. Genomic DNA was extracted from peripheral blood leukocytes according to standard methods. -420C>G polymorphism genotypes were determined by polymerase chain reaction followed by restriction analysis with 5 units of BbsI. Digested products were separated on 2.5% agarose gel with ethidium bromide staining.

Results: The frequencies of RETN -420G allele in MI group, and control group were 0.45 and 0.41, which are met with the Hardy-Weinberg equilibrium. Compared with controls, there was no significant differences in distribution of genotypes and allele frequencies of -420 C>G polymorphic site in MI patients and controls. In addition, there was no significant difference between the genotypes with lipid profiles.

Conclusion: Our data suggest that the RETN -420C>G polymorphism is not associated with an increased risk of MI in a Tunisian population.

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Study of arterial rigidity QAS
Evaluation by radio frequency of the quality of carotide rigidity on level in real-time, in patients with cardiovascular risk factors

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Objective : Evaluation by radio frequency in real-time, the quality of arterial rigidity in patients with cardiovascular risk factors.

Method: 3 measurements each carotid artery to determined coefficients, parameters rigidity, velocity of wave of pulse according by age. Quality of the rigidity of the arterial wall is measured from the velocity of the wave of pulse (PWV), variation of arterial diameter blood pressure on level of humeral artery, with measures of rigidity parameters.

Results: 2 carotid axes: 200 patients, 3 groups (age 30- 69) according by age, sex,race 2 groups: white race, dyslipidemia, type II diabetes, smoking: according by sex. 1 group: 60 M, white race, the 2 group: 40 F (white race), 3 group 50 patients (hypertension, overweight) according to the sex and race; in 2 sub-groups: group A 25 M (20 white race, 5 black) group B 25 F white race. Brachial blood pressure calculated to obtain the velocity of the wave of pulse, coefficients of arterial rigidity, local pressure. The pathological wave of pulse >13m/s, coefficients of rigidity: >11, ß > 20. In the 2 groups (10% men, 8% women) had pathological >11, ß>20 and velocity propagation of PWV >13 m/s. In 3 group 20% M, 12% F with hypertension, pathological rigidity, local pressure systolic increased despite the treatment.

Conclusion: QAS non invasive mesure of arterial rigidity on carotid, independent marker, in patients with cardiovascular risk factors, study the local and central blood pressure hypertensive patients to better control a pharmacological therapy. QAS by radiofrequency calculation of arterial rigidity on carotid level, parameters of rigidity , , and velocity of propagation of wave of pulse, the local and central pressure of the studied artery. In 1 and 2 group, patients with nicotinic intoxication and risk factors, in 3 group (hypertension, overweight) not find pathological data neither differences according to the sex and the race among all hypertensive patients. 2 patients of race black, 5 of the sub-group (hypertension) with pathological coefficient > 11.

QAS: calculation of arterial rigidity, RF: radio frequency, PWV (wave velocity pulse), , ß : stiffness parameters .

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High density lipoprotein-anionic peptide factor effect on reverse cholesterol transport in type 2 diabetic patients with and without coronary artery disease

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Objectives: To verify if HDL3 Anionic Peptide Factor (HDL3-APF) is as an apolipoprotein that promotes the reverse cholesterol transport.

Design and Methods: We investigated a possible association between plasma HDL3-APF concentration, cholesterol efflux from Fu5AH cells and cholesteryl ester transfer protein (CETP) activity in type 2 diabetic patients with coronary artery disease (CAD) (n=36), those without CAD (n=20), and 37 healthy subjects.

Results: Plasma APF concentrations were decreased in diabetics with CAD compared to controls ($p<0.01$). Cellular cholesterol efflux was decreased in diabetics without and with CAD, ($p<0.01$ and $p<0.001$ respectively). CETP activity was significantly elevated in all patient groups. Multiple linear regression analysis shows that cholesterol efflux was independently and positively related only to APF concentrations in whole population and controls.

Conclusions: APF is likely to be a key independent factor for promoting cellular cholesterol efflux in healthy subjects. However this association is altered in type 2 diabetes.

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Mediterranean diet decrease homocysteine levels and increase thiolactonase activities in elderly patients at high risk of cardiovascular disease

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Background: Food items might have a synergistic and antagonistic effect on health. The Mediterranean diet (MD) has long been associated with lower incidence of cardiovascular disease and cancer. Elevated blood homocysteine is a risk factor for cardiovascular disease. A 5-mmol/L increase is associated with an approximately 70% increase in relative risk of cardiovascular disease in adults. For patients with established risk factors, this risk is likely even greater. Thiolactonase; the antioxidant enzymatic component of HDL plays a crucial role in metabolizing homocysteine thiolactone and reducing homocysteine endothelial damages.

Objective: A total of 53 elderly coronary artery disease patient swere recruited and divided into diabetic patient (n= 27; age= 68.3 years) and non diabetics (n = 26; age= 68.7 years). We evaluated plasma homocysteine levels (tHcy); thiolactonase activity (HTase) and studied the effects of adherence to a Mediterranean diet on them.

Material & methods: Plasmatic tHcy was determined by capillaryGC-MS; HTLase activity was estimated by a kit immunoassay. Dietary intakes were evaluated by a validated food frequency questionnaire and transformed into a score traducing adherence to MD.

Results: Significantly higher median tHcy levels were found in diabetic patients as compared to the non diabetic ones (18.5 µmol/L vs. 5.8 ; p=0.057) associated to lower HTase activities (266.8U/L vs. 327.4; p=0.058). Moreover, in the diabetic patients tHcy levels were negatively associated with thiolactonase activities (r= -0.637; p 0.00). In this group, MD reduced diastolic blood pressure (r= -0.558; p=0.014). In the non diabetic group, tHcy levels were negatively correlated with HTase activities (r=0.759; p=0.00) and diet score (r= -0.0706; p=0.013) while HTase activities were positively correlated with diet score (r= 0.0759; p= 0.00).

Conclusion: Elevated homocysteine in diabetic patients may partly be explained by the diminished thiolactonase activities and could be considered as an additional risk factor for cardiovascular events. Adherence to MD could be the first efficient step to prevent these complications.